[17] CDC Launches New Antismoking Campaign

According to a press report, a new anti-smoking advertising campaign has been devised by the Centers for Disease Control and Prevention (CDC). The ads are apparently targeted toward nonsmokers in the workplace and were unveiled during a June 23, 1993, meeting of the Interagency Committee on Smoking and Health. Intended for television and other media, the CDC ads reportedly urge employees to tell their bosses they want a smoke-free workplace. *See Occupational Health & Safety News*, July 9, 1993.

MEDIA COVERAGE

[18] "Invironment: The New Horizon - Indoor Environmental Quality," *Consulting Specifying Engineer*, June 1993

This supplement to the monthly publication took an engineer's look at the effective construction of buildings which address total indoor environmental quality in their design. The article used the accommodation of smokers in the workplace as a case study for developing a model.

The article included strategies for accommodating smokers in high-rise, mid-rise, and small office buildings, which included technology such as displacement ventilation, air filtration technology, air pressure zones, and individual smoking areas.

[19] "The Great American Smokeout", CNN *Crossfire*, July 2, 1993

Crossfire hosts Mike Kinsley and Pat Buchanan discussed discrimination against smokers and smoking with guests Dave Brenton, editor of The American Smoker's Journal, and Dr. Sidney Wolfe, director of Public Citizen's Health Research Group. The lively, yet largely unsubstantial, debate yielded comments that a typical nonsmoker's exposure to ETS amounts to roughly the equivalent of one cigarette a year, and that airlines are in financial trouble in part because they banned smoking on planes.

[20] "How Secondhand Smoke Hurts Kids," R. Israeloff, *Parents Magazine*, August 1993

Citing the EPA Risk Assessment on ETS, this article discusses the purported effects of ETS exposure on young and unborn children. The alleged effects on young children are said to include increased incident of infections, asthma, increased chance of SIDS and lung cancer. The purported effects of ETS exposure to fetuses are alleged to include low birth weight, diminished lung function and a diminished milk volume in the mother.

The article goes on to offer some suggestions to lower children's exposure to ETS. The suggestions ranged from quitting smoking and finding smoke-free day care centers to joining local health and advocacy organizations that fight for the rights of nonsmokers.

[21] "Anti-scent Sentiment Catching on in Offices," S. Rostler, *The Plain Dealer*, July 16, 1993

The author of this article discusses the issue of fragrance bans in workplaces and compares them to ETS bans. The ban on fragrances at public meetings in San Francisco is mentioned, and a number of people who are annoyed by perfumes are quoted regarding their coping strategies when in the presence of heavy scents in the workplace. A partner in a Cleveland law firm specializing in occupational safety and health issues observes that there is no easy way for employers to enforce a ban on scents.

SCIENTIFIC/TECHNICAL ITEMS

LUNG CANCER

[22] "Environmental Tobacco Smoke and Lung Cancer," E.T.H. Fontham, P. Correa, P.A. Buffler, R. Greenberg, P. Reynolds, and A. Wu-Williams, *The Cancer Bulletin* 45(1): 92-94, 1993 [See Appendix A]

This brief article discusses the 1991 Fontham, et al., study of spousal smoking. In addition to providing a summary of the design and results of the study, the authors mention potential confounders. They claim that their calculated risk estimates "persisted" after adjustment for vegetable consumption, family history of lung cancer, and employment in "high-risk" jobs. They also indicate that they have collected some data on radon levels, and discount radon as a potential confounder, as well.

Cardiovascular Issues

[23] **"Association of Passive Smoking with Increased Coronary Heart Disease Risk Is Not Explained by Elevation of Leucocyte Count," M.S. Green, J. Shaham, J. Green, G. Harari, and J. Bernheim, *European Journal of Public Health* 3: 14-17, 1993 [See Appendix A]**

In this study, conducted in Israel, leucocyte counts are compared in smokers, nonsmokers and persons reportedly exposed to ETS. [Leucocytes are a kind of blood cell. Leucocyte count has been reported to be associated with an increased risk of coronary heart disease.] The authors report that reportedly ETS-exposed persons had leucocyte counts similar to those of nonsmokers.

Respiratory Diseases and Conditions — Adults

[24] **"Effects of Maternal Smoking and Childhood Respiratory Illness on Pulmonary Function in Young Adults: The CARDIA Study," J.E. Dunn, S. Kellie, K. Liu, and J. Keller, Society for Epidemiological Research, 1993 Annual Meeting, Keystone, Colorado, Abstract No. 306, June 1993 [See Appendix A]**

According to these authors, a comparison of current lung function data and reports of maternal smoking and childhood respiratory illness before age ten suggest that early lung infections and maternal smoking may "affect" adult lung function.

[25] **"Passive Smoking and Evolution of Lung Function in Young Adults. An Eight-Year Longitudinal Study," M.S. Jaakkola, J.J.K. Jaakkola, P. Ernst, and M.R. Becklake, *American Review of Respiratory Disease* 147(4 Part 2): A215, 1993 [See Appendix A]**

In this abstract, the authors report on a study in which 117 never smokers were tested for lung function at the start of the study and then again after eight years. ETS exposure was reportedly not associated with "clinically important ventilatory impairment," as measured by change in lung function over time.

[26] **"Effets Sanitaires d'une Exposition Chronique a la Fumee de Tabac sur une Population de Non Fumeurs (Health Effects of Chronic Exposure to Tobacco Smoke on a Non-Smoker Population)" [English abstract only], A.M. Laurent, A. Bevan, N. Chakroun, Y. Courtois, B. Valois, M. Roussel, B. Festy, and S. Pretet, *Rev. Pneumol. Clin.* 48: 65-70, 1993 [See Appendix A]**

In this study, conducted in a prison, 14 nonsmokers lived for one month in a cell with three smokers. At the end of the study, there were reportedly no differences in measurements of lung function, expired carbon monoxide, cotinine, or other possible indicators of exposure.

Respiratory Diseases and Conditions — Children

[27] **"Privilege and Health — What Is the Connection?" M. Angell, *New England Journal of Medicine* 329(2): 126-127, 1993 [See Appendix A]**

This editorial focuses on the correlation between socioeconomic status (usually measured using income, education and/or profession) and health endpoints. The author uses childhood asthma and "passive smoking" as an example of the need to consider socioeconomic status in interpreting a reported association.

[28] **"Is Passive Smoking a Cause of Asthma in Childhood?" R. Ehrlich, M. Kattan, and D.E. Lilienfeld, *Journal of Smoking-Related Disorders* 4(2): 91-99, 1993 [See Appendix A]**

Available studies investigating parental smoking and childhood asthma or lower respiratory conditions (e.g., wheezing) are reviewed in this paper. The authors acknowledge inconsistencies among the studies, and indicate that further questions remain. Nevertheless, in their opinion, a causal relationship between maternal smoking and childhood asthma is supported by the available data.

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ETS Exposure and Monitoring

[29] **"Measuring Exposure to Environmental Tobacco Smoke in Studies of Acute Health Effects," M.C. Marbury, S.K. Hammond, and N.J. Haley, *American Journal of Epidemiology* 137(10): 1089-1097, 1993 [See Appendix A]**

This paper reports on a study comparing questionnaires, ambient nicotine measurements, and urinary cotinine measurements as methods for characterizing ETS exposure in a population of 48 children under the age of two. The authors report that ambient nicotine measurements and urinary cotinine measurements provided better data than did questionnaires, but that the two measures were so closely correlated as to be redundant. As ambient nicotine measurements were more easily obtained, the authors suggest that they will use that method in their future work.

Indoor Air Quality

[30] **"Indoor Air: Potential Health Risks Related to Residential Wood Smoke, as Determined Under the Assumptions of the US EPA Risk Assessment Model," K.S. Sidhu, J.L. Hesse, and A.W. Bloomer, *Indoor Environment* 2: 92-97, 1993 [See Appendix A]**

The authors of this paper apply EPA methodology to generate risk estimates for components of wood smoke, and project excess cancer cases of 0 to 5 in a population of 100,000 for formaldehyde and 0 to 10 in a population of 100 million for polycyclic aromatic hydrocarbons. They suggest that exposure to wood smoke "should be recognized as a potentially significant health risk factor," especially for "sensitive populations," i.e., children, asthmatics and persons with heart disease.

[31] **"A Prevalence Study of the Sick Building Syndrome (SBS) and Facial Skin Symptoms in Office Workers," B. Stenberg, K.H. Mild, M. Sandstrom, J. Sundell, and S. Wall, *Indoor Air* 3: 71-81, 1993 [See Appendix A]**

In this paper, Swedish researchers report on a selected set of data collected in a study of nearly 5,000 office workers. They report that female gender, asthma/rhinitis, paperwork, and work with VDTs were related with an increased prevalence of sick building symptoms in their study population. They also report a relationship between perception of health and perception of indoor climate in their sample.

[32] **"Volatile Organic Compounds in Ventilating Air in Buildings at Different Sampling Points in the Buildings and Their Relationship with the Prevalence of Occupant Symptoms," J. Sundell, B. Andersson, K. Andersson, and T. Lindvall, *Indoor Air* 3: 82-93, 1993 [See Appendix A]**

The authors of this Swedish study report that occupant reports of symptoms were associated with elevated formaldehyde levels and with a decrease in total VOC levels between supply air and room air. They termed the latter "'lost' TVOC," and suggested it could be related to chemical transformations of the substances.

IN EUROPE & AROUND THE WORLD

REGULATORY AND LEGISLATIVE MATTERS

European Parliament

[33] **Briefing Paper on "Passive Smoking" Released**

On June 22, 1993, the Directorate General for Research of the European Parliament released a briefing paper entitled "Smoking Tobacco and Health: Passive Smoking." The paper provides an overview of governmental studies on ETS, including the EPA Risk Assessment on ETS, to support its position that "[t]he inhalation of other people's tobacco smoke has become recognised as a serious health hazard." In a cursory fashion, the paper links ETS exposure to lung cancer; irritation of eyes, nose, throat and lower respiratory tract and adult asthma; heart disease; exacerbation of sick building syndrome; and diseases in children such as bronchitis, pneumonia, reduced lung function, glue ear, asthma, wheezing, low birth weight and cot deaths.

The bulk of the document focuses upon legal issues. Strengths and weaknesses of ETS products liability cases are noted. Among the weaknesses discussed are

causation problems, lack of knowledge by the tobacco industry about alleged risks prior to the early 1980s, and intervening causation, i.e., smokers might not have heeded a warning about purported dangers of ETS exposure even if the tobacco companies had given it.

Cases against the industry and employers all over the world are discussed, including *TIA v. AFCO, Broin, Butler, Veronica Bland,* and *Liesel Scholem.* Richard Daynard of the Tobacco Products Liability Project is cited as a source of information regarding cases in the United States in which nonsmokers have recovered benefits or damages on the basis of a variety of legal theories. The paper concludes with a discussion of the granting of legal aid in the United Kingdom for a woman who is planning to sue her boss for injuries allegedly suffered as a result of ETS exposure. "The granting of legal aid in this case may show the direction in which the climate of opinion is moving," the paper states.

GERMANY

[34] Railway System Places Further Restrictions on Smoking

According to a press report, the Bundesbahn is planning to restrict smoking to seats in the smoking area of train carriages. Previously, smoking was also permitted in side aisles and entrances. The Ministry of Transport reportedly announced that the change would take place sometime this year. *See Die Tabak Zeitung,* May 21, 1993.

LEGAL ISSUES AND DEVELOPMENTS

AUSTRALIA

[35] Former Spouses Seek Injunctions to Stop Smoking in Front of Children

According to a press report, applications have been filed in Adelaide and Melbourne to stop former spouses from smoking in front of their children. The cases are believed to be the first of their kind in Australia and are expected to be heard later this month in Family Law Court. *See Canberra Times, Australian, Age, West Australian, Advertiser,* and *Courier Mail,* July 7, 1993.

[36] Employers Advised to Make Premises Smoke Free

Partners in the personal injury insurance division of Phillips Fox have reportedly advised employers to ban smoking in the workplace. "Employers must ensure that their premises become smoke-free or they face criminal and civil litigation," state partners John Boland and Norman Abrams. They apparently regard information about the alleged health effects of ETS as "unequivocal medical and scientific evidence." *See Age,* July 13, 1993.

OTHER DEVELOPMENTS

FRANCE

[37] Booklet Counters Misrepresentations About Smoking Bans

The Tobacco Documentation and Information Centre in Paris has issued a booklet entitled "What One Hears About Tobacco" to correct misrepresentations that have been made about public smoking bans in France and the European Community. Among those issues discussed are the claims that smoking is regulated in public places everywhere in Europe and that ETS endangers the health of nonsmokers. French legislation on smoking in public places is reprinted in the booklet.

NETHERLANDS

[38] Amsterdam Airport to Impose Smoking Restrictions

In 1994, Amsterdam's Schiphol will reportedly become the first airport in Europe to impose significant restrictions on smoking. With the exception of a few designated smoking areas, smoking will not be permitted in the departure and arrival halls and at the luggage carousels. The restrictions will apparently cost millions of guilders as ashtrays will need to be replaced with rubbish bins and no smoking signs. *See Algemeen Dagblad,* May 28, 1993.

UNITED KINGDOM

[39] Employees Forbidden from Smoking When in Uniform

The Pembrokeshire NHS Trust in West Wales has reportedly banned smoking for workers at anytime that they are in uniform, including while on or off duty. Apparently, those wishing to smoke on the way to or from work must change out of uniform to do so. The policy is believed to be the first of its kind in the country. According to a health workers union official, the policy goes too far. "We fully support no smoking in hospitals and health centers," he reportedly said, "but what employees do when they are off duty is up to them." *See Today*, July 2, 1993.

[40] *Smoking in Restaurants* Leaflet Produced

The Restaurateurs Association of Great Britain has produced a leaflet entitled *Smoking in Restaurants* for distribution throughout the leisure industry. The leaflet, which was produced in conjunction with the Tobacco Advisory Council, provides practical suggestions for accommodating smokers and nonsmokers in the same restaurant.

[41] Bare Majority Favors Restaurant Smoking Bans

According to a survey conducted by MORI for the Health Education Authority, some 51 percent of respondents reportedly think smoking should be banned in restaurants. Forty-five percent of those surveyed said there should be designated smoking areas, and three percent said customers should be free to smoke wherever they wish. The survey was apparently conducted last year, but the results have yet to be published. According to a press report, the findings contrast with current practice, as a large majority of restaurants permit patrons to smoke when and where they wish. *See The Times*, July 17, 1993.

APPENDIX A

The numbers assigned to the following article summaries correspond with the numbers assigned to the synopses of the articles in the text of this Report.

LUNG CANCER

[22] "Environmental Tobacco Smoke and Lung Cancer," E.T.H. Fontham, P. Correa, P.A. Buffler, R. Greenberg, P. Reynolds, and A. Wu-Williams, *The Cancer Bulletin* 45(1): 92-94, 1993

"In late 1985, a multicenter US study was initiated to evaluate the association of ETS with the risk of lung cancer in nonsmoking females. The study was designed to minimize some of the methodologic problems that have been discussed in review[s] of ETS lung cancer studies by the National Research Council and the International Agency for Cancer Research, among others. These included misclassification of smoking status, inaccuracy of case diagnosis and cell type, recall bias, ETS exposure from sources in addition to the spouse, and inadequate control of potential confounders. The findings of the first 3 years of this study are summarized here."

"The estimated risk of lung cancer in nonsmoking women that is associated with living with a spouse who smoked was approximately 30%, regardless of which control group was used in the comparison. An increase in the risk of approximately 50% was observed for adenocarcinoma of the lung compared to each control group. Separate analyses were conducted for subjects who personally responded and for those whose information was obtained from surrogate respondents. The findings were consistent for self- and proxy-respondents. All odds ratios were adjusted for age, race, geographic region, respondent type, income, and education. An approximate 30% risk of lung cancer associated with spousal ETS exposure persisted after an additional adjustment was made for the consumption of vegetables (the most significant food or nutrient factor), family history of lung cancer, and employment in high-risk occupations or industries. Household radon levels were determined in a sample of case and control homes under separate funding. Radon levels are quite low in all of the areas included in our study: <1% of all homes tested had levels of ≥4 pCi/L. The observed increased risk of lung cancer associated with ETS exposures is unlikely to result from confounding by radon, diet, or other such factors."

"The findings of this study — which included methods to evaluate recall bias, minimize misclassification of smokers as nonsmokers, ensure accuracy of diagnosis and classification of lung cancer, and adjust risk estimates for potential confounders — are consistent with and extend the findings of numerous published reports that did not address all of these issues. The overall 30% increased risk associated with ETS exposure from a smoking spouse is remarkably close to the 25% to 34% estimates of the evaluation of relevant studies in the 1986 report of the National Research Council. The significant positive dose response to exposure to tobacco smoke within households, in occupational settings, and in social settings during adult life strongly supports an etiologic role of ETS in lung cancer in nonsmokers and extends the findings from the home into the workplace and public settings."

CARDIOVASCULAR ISSUES

[23] "Association of Passive Smoking with Increased Coronary Heart Disease Risk Is 23Not Explained by Elevation of Leucocyte Count," M.S. Green, J. Shaham, J. Green, G. Harari, and J. Bernheim, *European Journal of Public Health* 3: 14-17, 1993

"The increased risk of coronary heart disease in cigarette smokers may be due at least partly to an elevation of the leucocyte count. Chronic passive smoking has also been found to be associated with an increased risk of coronary heart disease, but its effect on the leucocyte count has not been reported. in this study 250 male factory employees aged 20-64 years were interviewed on smoking behaviour and exposure to environmental tobacco smoke, and blood counts were determined. Urinary cotinine was measured by radio-immunoassay and corrected for urinary creatinine concentrations. Mean leucocyte count was significantly higher among smokers compared with nonsmokers. On the basis of smoking history, passive smokers had leucocyte counts similar to non-smokers."

"The aim of this study was to compare the leucocyte count in cigarette smokers with that of non-smokers exposed to varying concentrations of environmental tobacco smoke. Urinary cotinine concentrations were used to determine the extent of exposure to environmental tobacco smoke."

"These findings demonstrate that leucocyte count is not significantly elevated in non-smokers exposed to different levels of environmental tobacco smoke. In addition, among light smokers there was no increase in leucocyte count. This suggests that if in fact elevation of the leucocyte count contributes to the risk of coronary heart disease in smokers, this is not likely to be the mechanism for passive smokers or indeed for light smokers. In general the mechanism for the increased risk of coronary heart disease in passive smokers is not well understood. It has been postulated that it may be due to effects on blood factors such as platelet aggregation or through a role of the damaging and mutagenic effects of agents such as the polycyclic aromatic hydrocarbons on the endothelial and smooth muscle cells. In addition, extensive exposure to environmental tobacco smoke may result in reduced oxygen supply to the myocardium."

"The findings of this study suggest that at the population level, at least among adult men, chronic exposure to environmental tobacco smoke has little or no effect on leucocyte count."

"Despite this finding, an effect in some heavily exposed individuals cannot be excluded."

Respiratory Diseases and Conditions — Adults

[24] **"Effects of Maternal Smoking and Childhood Respiratory Illness on Pulmonary Function in Young Adults: The CARDIA Study," J.E. Dunn, S. Kellie, K. Liu, and J. Keller, Society for Epidemiological Research, 1993 Annual Meeting, Keystone, Colorado, Abstract No. 306, June 1993**

"The effects of maternal smoking and childhood respiratory illness (self-report of physician-confirmed asthma, pneumonia or bronchitis before age 10) on adult pulmonary function were assessed using the baseline (1985-1986) data of CARDIA (a study of lifestyle and cardiovascular disease risk factors in 5,155 Black (B) and White (W), male (M) and female (F) adults aged 18-30 years). Mean FEV1/FVC and FEF25-75 in exposed and unexposed groups were compared within each race, sex, and current smoking status stratum while controlling for age, height, and pack-years of cigarettes. Even when excluding ever-asthmatics, maternal smoking was associated with lower FEV1/FVC and FEF25-75 in BF and WF smokers. Childhood pneumonia or bronchitis was associated with lower FEV1/FVC in BM and WM non-smokers and WF smokers and WM non-smokers, all without history of asthma. Excluding those with active asthma, childhood asthma was associated with reduced FEV1/FVC in all non-smokers *except* WF, as well as in WM smokers, and with reduced FEF25-75 in all non-smokers *except* WM, as well as in BF and WM smokers. These data suggest that early lung infections and maternal smoking may affect adult lung function independently of asthma, and the effect of childhood asthma on adult lung function can be seen in persons without active asthma."

[25] **"Passive Smoking and Evolution of Lung Function in Young Adults. An Eight-Year Longitudinal Study," M.S. Jaakkola, J.J.K. Jaakkola, P. Ernst, and M.R. Becklake, *American Review of Respiratory Disease* 147(4 Part 2): A215, 1993**

"Little is known about the effects of passive smoking on lung function during young adulthood. Our objective was to examine the relation between exposure to environmental tobacco smoke (ETS) and the rate of change of ventilatory lung function in young adults during a study period of 8 years, with an additional aim to recognize susceptible subgroups. . . . There was no statistically significant relation between exposure to ETS during or before the study period and evolution of FEV_1 or FEF_{25-75}. . . . [A] physiologically relevant effect on evolution of ventilatory function is unlikely to be associated with the exposure levels experienced by our study population. A statistically significant but physiologically unimportant relation between cumulative home exposure to ETS before the study and [change in FEV_1] was observed in a subgroup of subjects 25 years of age or younger. There was no evidence of modification by atopy, wheezing or gender. Our results suggest that exposure to ETS in young adulthood in office work environment and at home does not lead to clinically important ventilatory impairment in the exposure levels as experienced in Canadian housing conditions in the 1980's. This does not refute the possibility that higher exposure levels may be harmful."

[26] **"Effets Sanitaires d'une Exposition Chronique a la Fumee de Tabac sur une Population de Non Fumeurs (Health Effects of Chronic Exposure to Tobacco Smoke on a Non-Smoker Population)"** [English abstract only], A.M. Laurent, A. Bevan, N. Chakroun, Y. Courtois, B. Valois, M. Roussel, B. Festy, and S. Pretet, *Rev. Pneumol. Clin.* 48: 65-70, 1993

"A study devised to evaluate the effects of chronic exposure to tobacco smoke on the health of a population of non-smokers was conducted in a prison."

"Fourteen volunteers among male, non-smoking prisonners [sic] in good health and without history of lung disease were put for thirty days in a cell that was already occupied by three smokers. These subjects were examined on arrival and on the 30th day of their imprisonment. The examination included filling a questionnaire concerning daily habits and food, respiratory function tests, measurement of CO in the expired air, measurement of nicotine, cotinine, thiocyanates and cadmium concentrations in blood and in urine, and measurement of mutagenic substances excreted. The purpose of the study was to detect possible changes in the variables tested between the first and last days of confinement."

"No significant difference could be demonstrated within this lapse of time. These results are in agreement with the data available in the literature. In the present state of our knowledge, it is difficult to find evidence of a biological impact, notably on the concentrations of the specific indicators nicotine and cotinine, in subjects passively exposed to tobacco smoke."

RESPIRATORY DISEASES AND CONDITIONS — CHILDREN

[27] **"Privilege and Health — What Is the Connection?"** M. Angell, *New England Journal of Medicine* 329(2): 126-127, 1993

"Anyone who follows the medical literature knows that 'socioeconomic status' is a powerful determinant of health. In current jargon, socioeconomic status refers to a mix of factors that shape a person's relative social advantage. It is usually gauged by income, education, profession, or some combination of the three, but no one knows exactly which factors determine health, much less how they do so. It does not seem to be simply a matter of the privileged having better access to health care. Nevertheless, in study after study socioeconomic status emerges as one of the most important influences on mortality and morbidity."

"So closely does socioeconomic status correlate with that health that it confounds the interpretation of much clinical research. For example, studies of the effect of passive smoking on childhood asthma are uninterpretable unless an attempt is made to control for socioeconomic status. Without such control, it is impossible to know whether the increased prevalence of asthma in the children of smokers is really because of passive smoking or because smokers are more likely to be poor and poverty itself is associated with a higher prevalence of asthma. . . . Indeed, if the direct effect of a variable under study — for example, passive smoking or exposure to lead — is small, and the effect of socioeconomic status is large, it may be very difficult to correct for socioeconomic status adequately. In such instances, stratifying subjects in only a few groups according to income or education may not be sufficient. To eliminate entirely the confounding effect of socioeconomic status may require stratifying subjects into a great many subgroups."

"Yet, despite the importance of socioeconomic status to health, no one knows quite how it operates. It is perhaps the most mysterious of the determinants of health. Income, education, and profession are not likely to influence health directly. Instead, these factors are almost certainly proxies for other variables that have a direct impact on health. But what are these variables? Most relevant studies attempt to control for such obvious ones as cigarette smoking and heavy alcohol consumption, both of which are more frequent among the disadvantaged. And the increased frequency of trauma and substance abuse among the poor cannot explain the increased morbidity and mortality from other causes. One can imagine a host of other influences — such as diet, stress, exposure to infectious agents or toxic chemicals — that are related to socioeconomic status, but there is very little evidence to point to any of them as a major cause of the health difference between the advantaged and the disadvantaged."

"Except for a few special conditions that affect black Americans disproportionately, such as hypertension, the poorer health of black Americans probably reflects

other correlates of lower socioeconomic status rather than race itself."

"[W]e are too quick to 'medicalize' many social ills. For example, we 'treat' drug abuse, as well as kleptomania, compulsive gambling, and a host of other 'co-dependencies.' . . . [I]t may be time to reverse this tendency to medicalize, and to be more ready to regard medical illness as the result — direct or indirect — of social factors. Certainly, there can be little doubt that research on the connection between health and socio-economic status — given the strength of this connection — would yield important information about the pathophysiology of disease."

"Dealing with the social causes of disease and responding to its medical effects are not mutually exclusive. We should do both. People already burdened by poverty and lack of education should not also carry a disproportionate share of illness."

[28] **"Is Passive Smoking a Cause of Asthma in Childhood?" R. Ehrlich, M. Kattan, and D.E. Lilienfeld, *Journal of Smoking-Related Disorders* 4(2): 91-99, 1993**

"The number of epidemiological studies of the association between passive smoking and asthma and wheezing in children is growing and the aim of this review is to re-examine the epidemiological evidence. Because of underdiagnosis of asthma and the difficulties of defining asthma for epidemiological purposes, wheezing as well as non-specific bronchial hyperresponsiveness are included as outcomes of interest, although they may lack some specificity for clinical asthma. For purposes of exposition, the epidemiological studies are divided according to the type of population studied, viz. general population studies and studies of asthmatics using health services. These in turn are further grouped according to study design."

"Despite inconsistency among the studies reviewed, a coherent pattern is emerging. In particular, studies which have been able to separate out maternal smoking have been more consistently positive than those that did not make this distinction. Further, among the studies which quantified maternal smoking, nearly all have been able to show some exposure-response relationship between maternal smoking and some measure of asthma or wheeze."

"In general, the evidence for an effect of passive smoking on wheezing and other lower respiratory illness is more consistent for children under two years of age than that for older children."

"Despite the emerging evidence concerning the importance of maternal smoking, variation remains among study findings, and this is likely to continue. Part of this variation between studies is due to the difficulties of measuring accurately the child's true dose of smoke exposure. Furthermore, ETS is a complex mixture of agents, any or a number of which may be implicated."

"The association between maternal smoking and asthma and wheezing illness in children satisfies a number of the criteria for causality. There is reasonable consistency among studies, an exposure-response relationship has been demonstrated and an appropriate temporal relationship established in prospective studies."

"In addition, the association has biological plausibility although the mechanism remains to be defined, and there is not strong evidence that confounding accounts for the observed increase in risk."

"From a public health perspective the impact of such a causal relationship is considerable. Assuming a relative risk of asthma due to maternal smoking of 1.5, after controlling for confounding, and a maternal smoking prevalence of 30%, an attributable proportion of 13% can be calculated. This is the proportion of asthma and persistent wheeze in childhood that could be prevented in the absence of maternal smoking. This should add further weight to public health, clinical and educational efforts to reduce the burden of illhealth [sic] imposed on young children by the tobacco smoking habit."

ETS Exposure and Monitoring

[29] **"Measuring Exposure to Environmental Tobacco Smoke in Studies of Acute Health Effects," M.C. Marbury, S.K. Hammond, and N.J. Haley, *American Journal of Epidemiology* 137(10): 1089-1097, 1993**

"In preparation for an investigation of environmental tobacco smoke and lower respiratory illness in children under 2 years of age, we conducted a study to compare methods of characterizing exposure to environmental tobacco smoke, including questionnaires, urinary

cotinine measurement, and ambient nicotine measurement. Our purposes were to examine the relations among the three measures and their variability over time and to assess feasibility and logistic issues. Although ventilation rates were not measured, the study was conducted during a period when rates were expected to be stable."

"Thirteen girls and 35 boys under 2 years of age, all non-Hispanic white, were enrolled in the study. In 23 homes neither parent smoked; five homes were in the low exposure group."

"All three measured varied significantly, depending on which parent(s) smoked. Concentrations of ambient nicotine and urinary cotinine were higher when the mother smoked than when the father smoked and highest when both parents smoked."

"In this study, we initially compared three different methods of estimating exposure, questionnaire, urinary cotinine, and ambient nicotine, on a cross-sectional basis in a population with varying degrees of exposure. Urinary cotinine and ambient nicotine concentrations were highly correlated with each other. This correlation suggests that, even though one is a measure of exposure and the other a measure of dose, they provide equally valid but redundant information about exposure in this study population."

"Given equal validity, the choice between the two measures then depends on feasibility. One component of feasibility is the variability of the measure, as greater variability requires that more measurements be made for accurate exposure estimation. While both measurements showed variability over time, urine cotinine was more variable."

"[W]e found that one measurement of ambient nicotine during an 8-week period, when ventilation rates are relatively stable, would be adequate for achieving reasonable precision in our exposure estimate. In contrast, three measures of urinary cotinine would be needed to obtain the same degree of precision. In addition, we were able to collect complete data on ambient nicotine measurements, whereas we were unable to obtain urine samples at 20 percent of the visits. Thus, for this study population, air monitoring of nicotine is more feasible."

"[Q]uestionnaires will undoubtedly continue to be used in studies of environmental tobacco smoke, either as the whole or as a part of the assessment strategy. When used in conjunction with an objective marker, each can be used to help interpret the other. For example, in the cross-sectional study, both the pattern of activity room nicotine concentrations and urinary cotinine levels indicated that fathers smoked fewer cigarettes in the house than mothers, although fathers were reported to have smoked more. This was not due to a difference in where the fathers smoked, as all fathers were reported to have smoked in the living room or family room."

"[T]he pilot study provided information on the use of nicotine samplers. Placing nicotine samplers in both the activity room and the bedroom is unnecessary. The concentrations in the two rooms were highly correlated throughout the study."

Indoor Air Quality

[30] **"Indoor Air: Potential Health Risks Related to Residential Wood Smoke, as Determined Under the Assumptions of the US EPA Risk Assessment Model," K.S. Sidhu, J.L. Hesse, and A.W. Bloomer, *Indoor Environment* 2: 92-97, 1993**

"Residential wood smoke contains inorganic and organic particulates, ash, carbon monoxide, nitric oxide, nitrogen dioxide, formaldehyde, polycyclic aromatic hydrocarbons (benzo[a]pyrene, benzo[k]fluoranthene, benzo[b]fluoranthene, benzo[ghi]perylene, and indeno[1,2,3-cd]pyrene), phenols and sulfur dioxide. Benzo[a]pyrene and formaldehyde are classified as group B_2 and group B_1 carcinogens, respectively, by the United States Environmental Protection Agency. Reported concentrations and potential health risks of the released contaminants are discussed. In general, wood smoke is an irritant to the eyes, nose, and the respiratory tract. It has the potential to cause chronic respiratory problems. Because there are indications that wood smoke may cause adverse effects on human health, exposure to this source of air pollution should be minimized."

"The objectives of this paper are to formulate cancer risk assessments and to review the potential health effects of several indoor air contaminants released by wood-burning stoves."

"The exposure assessment and risk characterization for formaldehyde released from non-airtight wood-

burning stoves indicate that the number of excess cancer cases may vary from 0 to 5 in a human population of 100,000."

"The lifetime risk characterization showed that exposure to benzo[a]pyrene released by airtight wood-burning stoves may produce 0 to 10 excess cases of cancer in a population of 100 million people. It is an extremely small risk that falls well within what is considered acceptable."

"The concentration of individual contaminants released in wood smoke may be too low to cause any potential adverse health effect in the general population; however, chronic exposure to these contaminants can lead to respiratory problems, especially in sensitive populations such as children, asthmatics, and persons with cardiovascular disease."

"Although there is a variety of potential health risks from wood smoke, burning of wood is not presently regulated in many states of the United States. Exposure to this source of indoor air pollution should be recognized as a potentially significant health risk factor, especially for sensitive segments of the population. There is a need to minimize this source of indoor air pollution."

[31] **"A Prevalence Study of the Sick Building Syndrome (SBS) and Facial Skin Symptoms in Office Workers," B. Stenberg, K.H. Mild, M. Sandstrom, J. Sundell, and S. Wall, *Indoor Air* 3: 71-81, 1993**

"The aims of the present study among office workers were:"

"to determine the prevalence of perceived SBS symptoms (including skin symptoms) and the association between their occurrence and personal factors such as gender, age, smoking habits and atopy;"

"to describe perceptions of psychosocial factors and indoor climate at work;"

"to assess the relation between symptoms and psychosocial factors and exposure factors of relevance for the indoor climate at work and at home."

"The data analyzed are extensive and the results will be published in a series of papers with focus on different issues. This paper presents an overview of the office environment and the load of perceived symptoms as reported by the workers. It presents patterns and, in selected instances, details. The rationale is not mere space but also the fact that relations based on questionnaire data cannot be compared in detail with other questionnaire data. Differences in methods and phrasing affect the relations. Strong relation patterns, however, may be generally applicable. In a paper specifically addressing the male/female contrast, more detailed information about the distribution of the determinants and their relation to perceived symptoms are presented."

"At work the prevalence of complaints concerning physical factors was about five times higher than that for dwellings. Dry and stuffy air was by far the most common complaint in both sexes. . . . While draughts and too low a temperature were common complaints among females, too high a temperature was a common complaint among males. The indoor climate index was higher among females."

"Men more often reported interesting and stimulating work as well as a greater opportunity to influence work conditions. They also reported a greater workload."

"All symptoms, apart from difficulty in concentrating and scaling/itching scalp, were more prevalent among females. Eye, throat, and nose symptoms, feeling heavy-headed and facial skin complaints were the symptoms most commonly attributed to indoor climate factors."

"In the study population the prevalence of SBS-cases was 4% among men and 12% among women. The OR for being an SBS-case was 3.4 (95% CI: 2.7-4.2) for females compared to males. SBS-cases, general and skin symptoms were more prevalent under fifty years of age; among females SBS-cases were most prevalent under forty."

"Smoking was not associated with any increase in symptom indices whereas asthma/rhinitis entailed an increment of all symptom indices."

"The results from this study yielded complex patterns and relationships. Most prominent was the discrepancy in perceptions between males and females. This difference in perceived health could not be explained by the personal and predisposing factors dealt with in this study. For example asthma/rhinitis, an important determinant for symptoms, was distributed quite uniformly among males and females. Effect modification was observed for VDT work and skin symptoms

in the sense that prevalence increment was greater among males. This may be explained by the lower overall prevalence of skin symptoms among males, leaving substantial room for reaction to exposure compared to females."

"Dissimilar perceptions of physical climate indicate inequalities regarding indoor climate in which males and females live and work. The main risk indicators for symptoms were also associated with perceptions of a bad indoor climate. This supports the hypothesized connection between perception of health and perception of climate."

"Female gender, asthma/rhinitis, VDT and paper work and psychosocial factors are important determinants for symptoms compatible with SBS, including skin symptoms. Their importance as risk indicators was established by stratified assessments of exposure-response relationships. Even though the background for these associations is complex, the results imply that females and males in offices work under unequal conditions as indicated by different perceptions of physical and psychosocial factors."

"The implications of female gender as a risk indicator is [sic] an important issue that should receive further analysis. In that connection both a biomedical and a sociomedical perspective is essential. This study, focusing on non-building risk indicators, is supplemented by case-referent studies with emphasis on somatic, sociological and physical work place factors."

[32] **"Volatile Organic Compounds in Ventilating Air in Buildings at Different Sampling Points in the Buildings and Their Relationship with the Prevalence of Occupant Symptoms," J. Sundell, B. Andersson, K. Andersson, and T. Lindvall, *Indoor Air* 3: 82-93, 1993**

"Total Volatile organic compounds (TVOC) are determined in intake, room-supply and room air in 86 office rooms in 29 office buildings in northern Sweden. Measurements of formaldehyde were also made in room air. Building and room characteristics were identified and symptom reports collected from 1087 office workers."

"The aims of this investigation were to study changes in TVOC concentration from outdoor air to room air and to study the associations between symptoms reports and concentrations of TVOC and formaldehyde in room air."

"The occupant symptoms asked for in the questionnaire comprised three groups: (1) general symptoms including 'fatigue', 'feeling heavy-headed', 'headache', 'nausea/dizziness' and 'difficulties in concentrating'; (2) mucous-membrane symptoms including 'itching, burning or irritation of the eyes', 'irritated, stuffy or runny nose', 'hoarse or dry throat' and 'cough'; (3) skin symptoms, including 'dry facial skin', 'flushed facial skin' and 'itchy, stinging, tight or burning sensations in facial skin', 'scaling, itching scalp or ears' and 'hands dry, itching red skin'."

"The TVOC concentrations in the intake air and the supply air were generally higher than the concentration in the room air."

"[T]he more TVOC that are 'lost' in the room, the higher the concentration of formaldehyde. The meaning of the expression 'lost TVOC' in the room is that the concentration of TVOC is higher in the room supply air than in the room air. Likewise 'lost TVOC' in the ventilation system means that the concentration is higher in the intake air than in the room supply air."

"Buildings with a high prevalence of SBS had significantly lower TVOC concentrations in room air and a greater 'loss' of TVOC from intake to room air than buildings with a low prevalence of SBS."

"[F]ormaldehyde levels above the median value of 31 ug/m^3 acted as a significant risk indicator of mucous-membrane symptoms 'each week', of skin symptoms 'each week', and of 'at least one symptom each week'."

"'Loss' of TVOC from intake air to room air tended to be associated, though not significantly, with all types of symptom groups."

"The results of the present study are intriguing as they suggest that typically high TVOC concentrations in room air are not a positive risk factor of occupant symptoms. Instead, 'lost' TVOC and, to a lesser degree, formaldehyde act as risk indicators. In general, all the various analyses made show similar results. Important confounding factors, such as sex or work characteristics, were tested through stratification, multiple regression and logistic regression and were not found to change the general results. . . . The formaldehyde levels measured are all below the WHO guideline

value for the general population but above the value recommended for specially sensitive groups."

"A crucial question is, What might be the fate of the 'lost' TVOC? Up to now, interest in VOC has been directed towards source strength, dilution, dispersion, sorption and deposition but not transformations."

"The surprising covariation shown in the present study between symptom reports and TVOC demonstrates that there may not be a single monotonous dose-response relationship, at least not at low levels of exposure. The transformation of TVOC and single organic compounds in indoor air should be more extensively studied as well as their interaction with the physical and chemical microenvironments."

"Transformation products of TVOC are suspected of being a partial cause of occupant building-related symptoms."
